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THE BRITISH JOURNAL

OF

OPHTHALMOLOGY

MARCH, 1943

COMMUNICATIONS

A NOTE ON SOME SYMPTOMS ASSOCIATED WITH A RETINAL LESION

BY

E. G. HILL, M.Sc., PH.D., F.INST.P.

Introduction

THE purpose of this note is to record some subjective symptoms experienced by the writer since 1940 when abnormalities of vision were first noticed. Up to that time, when I was aged 47 years, vision had been good in both eyes. One year previously, on an occasion when the usual correction for reading was prescribed, both fundi were found to be normal.

In 1940 I began to experience some difficulties in vision accompanied by the appearance of a veiling patch of light. The eyes were then examined by Mr. Williamson-Noble who reported as follows: " I saw Dr. Hill first in November, 1940, when he told me that the right eye had appeared to be different from the left eye for the last five or six weeks. Vision, with correction of a small error of refraction, was 6/5 in each eye. Examination on the screen failed to show the presence of any scotoma. Ophthalmoscopic examination showed that the left fundus was normal, but in the right eye there was a shimmering area in the macular region, which I took to be located in the internal limiting membrane, and to be due possibly to some thickening of this. I have seen two other cases of a similar character in which there

was no material diminution of visual acuity and no progressive deterioration."

As the condition seemed somewhat rare the writer undertook to record details of any symptoms as far as they were accessible to subjective examination.

Further examination by Mr. Williamson-Noble in January, 1941, and October, 1942, confirmed the expectation that the appearances would remain unchanged and the vision was still found to be 6/5 partly with correction. Moreover, the complex of symptoms had, in the interval, shown no signs of alteration.

Account of the Symptoms

These subjective effects may conveniently be classified as follows: (*a*) Image distortion with localised aniseikonia, (*b*) the appearance of a luminous or a shadowy veil, (*c*) a phenomenon termed the " After Flash," (*d*) a convergence effect, and (*e*) pain. These effects will be treated separately.

(*a*) Image distortion.—The images of objects subtending angles up to a few degrees are given barrel-shaped distortion in the right eye which amounts to about 10 per cent. aniseikonia with irregularities within the apparently expanded area. Fig. 1 is a sketch of the appearance of a uniformly ruled objective field, the centre of which is a fixation point.

This apparent aniseikonia gives rise to disturbances of stereoscopic vision over this region, but the effect does not obtrude itself significantly in the ordinary use of the eyes. As, however, the area affected includes the foveal region, there is some difficulty with image fusion, and it is surmised that the strain involved accounts for the pain mentioned in (*e*) above. The aniseikonia can be made obvious with increase in apparent object size when vision is alternated between the two eyes, the right eye being opened as the left is closed.

Some points of interest may be mentioned in connection with the appearance of retinal projection when a uniformly illuminated field is scanned circularly with a pinhole aperture. The appearance is normal in the left eye, but the projected vascular system of the right eye is much more pronounced and is very irregularly shaped, having the central clear area contracted in size. The linear value of this contraction is of the order of 10 per cent., which conforms with the increase in image dimensions shown in Fig. 1. It may be deduced that the central retinal elements are bunched to this extent. The vascular system is so easily seen by this scanning process that it can be observed by half blinking the eye. It is thus possible to see simultaneously the vascular projection and, if the field is bright enough, the individual corpuscles traversing

it. These small silvery specks, which are visible in the normal eye against the sky brightness and seem usually to follow random paths, are here seen to be constrained to the vascular tracks, and

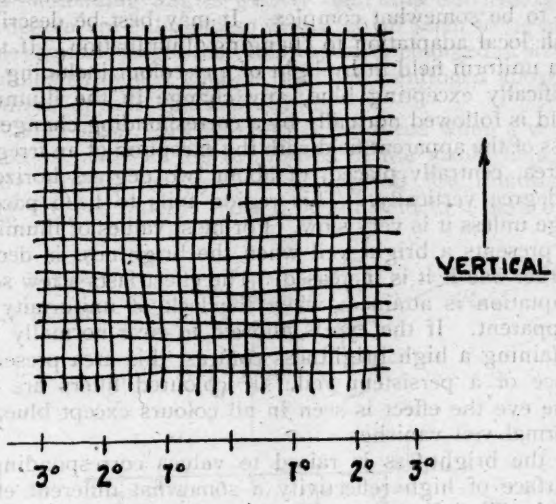

FIG 1.

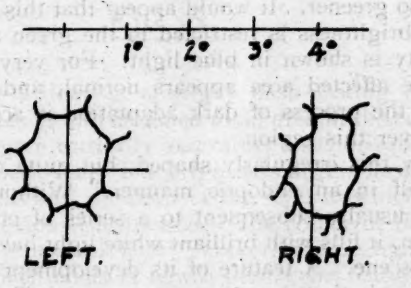

FIG 2.

it is possible to count the corpuscles traversing any particular vessel. This has been found to average about one per second under normal quiescent conditions.

Superposed on this projected vascular system there is a darker irregularly shaped area coinciding with the veiling effect to be

described. Fig. 2 is a sketch of the centre of the projected appearance resulting from a scanning pinhole. It shows also the comparison with the left eye.

(*b*) Luminous Veil.—The description of this effect seems of necessity to be somewhat complex. It may best be described as a sluggish local adaptation to changing illumination. If the eye looks at a uniform field lit by light of any colour including white, but specifically excepting blue, any change in the illumination of the field is followed normally by a corresponding change in the brightness of the apparent field with the exception of an irregularly shaped area, centrally placed, of about two degrees horizontally by one degree vertically. This region fails to keep pace with the change unless it is very slow. For most values of illumination the area presents a bright veil when the brightness is decreased and a darker one if it is increased. The effect lasts a few seconds until adaptation is attained, when the lack of uniformity is no longer apparent. If the eye is allowed to rove normally over a field containing a high brightness contrast this area presents the appearance of a persistent veil. If coloured filters are placed before the eye the effect is seen in all colours except blue, when the abnormal veil vanishes.

When the brightness is raised to values corresponding to a sunlit surface of high reflectivity a somewhat different effect is manifested. The affected area becomes and remains brighter than the surrounding field and all detail is lost in a localised glare. There is also some chromatic differentiation, as the area which is brighter is also greener. It would appear that this failure to limit the apparent brightness is restricted to the green and yellow, as no abnormality is shown in blue light. For very low values of brightness the affected area appears normal, and there is some evidence that the process of dark adaptation is accelerated in its later stages over this region.

Occasionally this irregularly shaped, but quite definite, region manifests itself in an endoptic manner. Without any intense lighting, but usually subsequent to a series of changing values of illumination, it fills with brilliant white light having no relation to the visual scene. A feature of its development which may be worth recording is that the area seems to grow like the spreading of a liquid when poured on a dry surface. It reaches its full brilliance in a matter of minutes, becomes entirely endoptic, and is therefore finally superposed on the vision of both eyes. At this stage the real field is blotted out unless it is very bright, but the effect now begins to fade, becoming pure green before it disappears. It may be emphasised that the presentation differs in every respect from migraine which has been previously experienced.

If the eye looks at bright sources on a dark field the apparent

size of the source differs in the two eyes. The distortion depends on the angular size roughly as follows: Sources subtending angles less than about ten minutes are the same in both eyes; sources subtending angles greater than this but less than about half a degree are enlarged so that they all seem to be about half a degree; sources larger than this appear unchanged in size. There is a similar chromatic change like that mentioned above. Small red sources appear greenish white.

(*c*) "After flash."—This effect is produced when a very abrupt reduction is made in the brightness of the visual field, such as suddenly closing the eyes, switching off the illumination, or averting the eye quickly from a bright field to a dark one. The

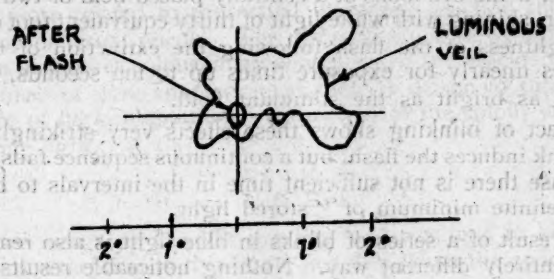

FIG 3.

result of these actions is a bright flash lasting approximately one-fifth of a second and confined to an area in the centre of vision about half a degree vertically and rather less horizontally. It may coincide with the fovea. The brightness of this flash is a function of the brightness and duration of the field to which the eye was immediately previously subjected. The colour of the flash is the same as the "stimulant field" except in blue light, when it is absent. This flash effect is now a permanent feature of vision in the affected eye and is superposed on the veil effect previously described. Fig. 3 shows the location and size of these areas.

It would appear that there is here some mechanism which can function as a light storage, but investigation has shown that although the flash is confined to the foveal area, this does not seem to be the region responsible for "storing the light" which is subsequently emitted. This has been found by varying the size of the bright stimulated area. For any given illumination the brightness of the after flash increases with angular increase of

the luminous field up to a few degrees diameter, beyond which no further effect is produced. On the other hand, if the luminous field extends all over the retina, but contains a dark centre of a few degrees diameter, no flash is experienced. It may therefore be concluded that the region responsible is confined to the retinal lesion.

Within limits of brightness, ranging from about five equivalent foot candles to sky brightness at midday, the brilliance of the after flash is, within limits, proportional to the product of the brightness and duration of exposure to the stimulant field. There is a saturation value which is attained, as far as can be estimated, when the flash becomes as bright as the previous field. As an example, if the eye looks at a centrally placed field of two degrees diameter, shining with white light of thirty equivalent foot candles, the brightness of the flash following the extinction of the field increases linearly for exposure times up to ten seconds, when it appears as bright as the stimulant field.

The act of blinking shows these effects very strikingly. The first blink induces the flash, but a continuous sequence fails to show it because there is not sufficient time in the intervals to build up some definite minimum of "stored light."

The result of a series of blinks in blue light is also remarkable in an entirely different way. Nothing noticeable results from a single blink even if preceded by protracted illumination, but a continuous sequence of blinks gives rise to the presentation of a very dark area coincident with the bright flash in other colours. This dark area deepens and persists throughout the sequence of blinks. The effect is not entirely absent in the other eye and there is some suggestion that it is an exaggeration of a normal effect.

(*d*) Convergence Effect.—Rapid movement of the eyeball or any attempt to converge the eye beyond the normal range of accommodation disturbs the apparent brightness of the field. This occurs over areas of the retina well outside those associated with all the previously described effects. If the affected eye looks at a large uniformly lit field and then converges, the uniform brightness of the apparent field is disturbed over a large area. The central region of a few degrees diameter darkens, while brighter areas appear outside it in a roughly concentric array. The peripheral region is not affected. Rapid movement of the eye causes this disturbed distribution to show momentarily. The effect disappears instantly with the relaxation of the convergence or cessation of rapid movement. There is no differentiation with colour and there is no colour change in the field. No effect is observable at low illuminations or in darkness.

Suggestions in the Direction of Explanation

The image distortion may presumably be attributed to a displacement of the retinal elements, and the character of the change shows that the centre of the retina must be bunched. The convergence effect seems to indicate a residual lack of rigidity in the posterior structure of the eye. It may also be mentioned here that, associated with these changes, a blind patch of ten minutes subtense made its appearance and is located, presumably significantly, exactly at the centre of the fovea.

The flash and veil phenomena are presumably bound up with the bleaching of the visual purple, and it would seem that the lesion has disturbed this mechanism over the affected area. As there is no reduction in the level of dark adaptation, the failure seems more related to the actual bleaching reaction than to the secretion of visual purple. The discrimination between the effects in blue and other colours may contribute to the elucidation of the mechanism of dark adaptation, as there is reason to suppose that the rate of dark adaptation is a function of the colour of the pre-adapting light.

Left Eye

In the foregoing the left eye has been assumed to be normal. The only exception to this is the fact that when the endoptic pool of light makes its appearance, there is some preliminary contribution ascribable to the left eye. This is established by the appearance of smaller patches of light, always the same shape, but visible only in the left eye for a short while before the effect becomes endoptic. None of the other appearances has ever been seen in the left eye.

Associated General Conditions

No reason can be adduced with any assurance for the retinal lesion which is presumably responsible for these abnormalities. The writer worked for many years with strong sources of radiation which, while screened from visible light, were found to transmit freely in the near infra-red region. Other co-workers have, however, not been affected in this way. It does appear, on consideration, that there may be some risk in looking at powerful sources through such filters, because the pupillary contraction normally associated with these sources will not be stimulated and the pupil will remain fully dilated. It may be pointed out that the residual radiation is of short enough wave-length to be transmitted, without significant loss, through cornea, lens and humours, and will therefore be focused as a powerful radiant,

invisible image on the fovea. Although there is no evidence that this has been a predisposing cause in the writer's case, unless the centrally placed blind patch be so considered, this may be a suitable occasion to issue a caveat to those engaged in occupations involving conditions similar to those described above.

It may be added finally that a complete dental radiological examination and a pathological blood test failed to reveal any contributory abnormalities.

A CASE OF BILATERAL INTERSTITIAL KERATITIS LEADING TO BLINDNESS

BY

Surg. Lieut.-Comdr. J. D. J. FREEMAN, R.N.V.R.

GLOUCESTER

THIS young stoker completely lost his sight within the space of six months. He was aged nineteen years at the time. There was a " blitz " on Bristol on the night of April 24, 1941, and a bomb fell close to where he was stationed. He did not actually remember anything striking his left eye, but there was a " very bright flash." His head ached after this and during the night his eye became very painful, so that he got very little sleep. The following morning he found his eye was inflamed, he reported at the sick-bay and was admitted to the Naval Hospital at Barrow Gurney the same day.

On admission he was found to have commencing acute interstitial keratitis of his left eye. There was a small epaulette up and out with a " ground glass " area of cornea below. He was treated with atropine and hot bathings and the pupil dilated well. The right eye seemed quite healthy and normal with 6/6 vision. There were no signs of disseminated choroiditis in either eye. The Wassermann reaction of his blood came back as full positive. On May 2 he had to be evacuated to an E.M.S. Hospital, together with most of the other patients in the hospital. He returned to the Naval Hospital on June 10. His left eye was now a shrunken blind eye. There was some panophthalmitis and the cornea had evidently perforated. While in the E.M.S. Hospital he had had a course of five injections of N.A.B. rising from 0·15 gm. to 0·60 gm. and three injections of Bismuth of 0·2 gm. The right eye was white and the cornea was clear. On July 6 the remains of the left eye were excised under general anaesthesia. The right gave no trouble and was white and free from any inflammation

until July 14. On this day he complained of pain and photophobia, and on examination was found to have ciliary congestion, and a ground glass patch in the cornea below. He was treated with atropine and hot bathing. The pupil was well dilated but the condition slowly advanced. On July 28 he had well-marked epaulettes above and below. The condition continued to advance and on August 8 the epaulettes were more marked above and below. There was now a mass of K.P. and white necrotic material on Descemet's membrane. As a result of this the iris was not visible. On August 25 he had only P.L. in the eye. The epaulettes had now become confluent and surrounded and diminished the area of the cornea. The cornea was uniformly opaque and the ocular tension was soft. On September 13 he was taken to London and seen in consultation by Sir Arnold Lawson, Consultant Oculist to the Navy. At Sir Arnold Lawson's suggestion, the blood sedimentation rate, complete differential blood count and X-ray of chest were done. These investigations all proved entirely negative. A course of old bovine tuberculin injections was also suggested and given. Prior to this, a course of M. & B. 693 had been given, followed by Pil Hyd. with Creta grs. 1 t.d.s., and later again Mist. Pot. Iod. grs. 5 t.d.s.—but all without avail. His general condition remained good throughout.

There was no previous history of any eye trouble. His mother and father were alive and well. He was the eldest of five children—two brothers and two sisters; none had died. One sister had a discharging ear when a baby and was rather deaf now. Otherwise family history was quite negative. He had had measles, chicken-pox and whooping cough and had had his tonsils removed. There was no history of head injury, bed-wetting or sleep walking; no convulsions, fits or faints. He went to an ordinary school until he was fourteen, leaving in the top class. He admitted he was not a good scholar. He was examined by Dr. MacDonald Critchley, Consultant Neurologist to the Navy, on September 19, 1941. He reported that physical examination revealed few signs. The arm jerks were a little brisker on the left side and the right ankle jerk was either very sluggish or absent. There were no stigmata of syphilis with the exception that his teeth were small, widely spaced, possibly peg shaped. At his suggestion a lumbar puncture was done and the cerebro-spinal fluid examined. This revealed no pathological features, and the Wassermann and Lange reactions were negative.

This boy has been granted a full pension and is now at St. Dunstan's, where he is doing very well. This is evidently a case of very severe congenital specific infection of his eyes starting as interstitial keratitis, and going on to bilateral gummatous infiltration of the uveal tract and loss of both eyes. The possibility

of a superadded tubercular infection was investigated, but no evidence was found of this. An exhaustive search of the literature has failed to reveal the recording of a similar case. For this reason it seemed worth recording this example.

LUMBAR PUNCTURE (September 23, 1941)

C.S.F. pressure, 14 Omm. Lab. Report on C.S.F.

Cells	26 per mm.
Globulin	not increased
Protein	40 mgms. per 100
Lange	1. 2. 3. 4. 2. 1. 1. 0. 0. 0.
W.R.	Negative

Blood Sedimentation Rate (Gibson) September 16, 1941.

Normal 94 per cent.

DIFFERENTIAL BLOOD COUNT (September 16, 1941)

Leucocytes	8'125 per c.mm.
Polymorphs	42'0 per cent.
Lymphocytes...	50'0 per cent.
Monocytes	2'0 per cent.
Eosinophils	5'0 per cent.
Basophils	1'0 per cent.

WEIGHT CHART

June 6, 1941	144 lbs.
June 27, 1941	144 lbs.
July 4, 1941	140 lbs.
July 18, 1941	140 lbs.
July 25, 1941	136 lbs.
August 1, 1941	137 lbs.
August 18, 1941	140 lbs.
September 2, 1941	142 lbs.
September 12, 1941	144 lbs.
October 3, 1941	154 lbs.

X-RAY EXAMINATION OF CHEST, September 16, 1941.

No abnormality shown.

ACNE ROSACEA KERATITIS AND RIBOFLAVINE. (VITAMIN B2)

BY

WINIFREDE M. FISH

OXFORD

IN a paper on "Rosacea Keratitis and Conditions with Vascularisation of the Cornea"[1] it is stated by Johnson and Eckardt that "thirty-six patients with rosacea keratitis were treated by the addition of riboflavine to the diet. Of these, nine also showed some cutaneous type of rosacea. Thirty-two of these patients had prompt healing of the corneal lesions"—usually within four days. "Of the thirty-six patients studied, only two gave a history of having eaten adequate amounts of egg white, liver and milk." It is inferred by the authors that "rosacea keratitis (so called) may be the direct result of deficiency of riboflavine."

In a subsequent communication[2] Johnson, referring to a paper by Sydenstricker and others on "The Ocular Manifestations of Ariboflavinosis,"[3] says that "changes in the cornea are described which appear to be identical with rosacea keratitis," and that "the term 'dietary keratitis' (used by Sydenstricker) appears to initiate a new designation of the condition familiar to ophthalmologists as rosacea keratitis, so called."

This seemed rather surprising, in view of the fact that in the Southern States of America, where riboflavine deficiency is common, acne rosacea is a rare disease and is not even mentioned in Sydenstricker's paper.

A series of forty-five cases of acne rosacea were therefore examined with the slit-lamp in order to find out whether they showed the type of corneal vascularisation demonstrated to the present writer by Sydenstricker as diagnostic of ariboflavinosis, *i.e.*, a bilateral, symmetrical, superficial proliferation of new streamer-like fine vessels from the limbal loops, forming a more or less regular pattern all round the cornea and extending towards its centre.

Of these forty-five cases all had the cutaneous lesions, and all but two had the corneal lesions typical of rosacea, but only three had bilateral corneal involvement, so that in forty cases there was a control eye uncomplicated by ulceration in which to look for the corneal vascularisation typical of ariboflavinosis, which, though not always equal, is always bilateral.

This type of vascularisation was absent in all forty cases.

Ten of these patients were treated with riboflavine alone in the dosage prescribed by Johnson and Eckardt, no local treatment being given, and—probably owing to the deprivation of atropine

under test conditions—these patients became so much worse within a week that routine treatment consisting of atropine, dark glasses, sulpho-calamine lotion (for the skin) and dilute hydrochloric acid (to be sipped with meals) had to be added to the vitamin B2 therapy, after which they, and twenty-three other cases similarly treated, healed within varying periods.

Ten other cases healed on routine treatment without riboflavine in about the same time.

One case (No. 1 (*b*)), with cutaneous rosacea but without corneal involvement of any kind, was untreated.

ANALYSIS OF 45 CASES OF ROSACEA

	Bilateral Keratitis	Unilateral Keratitis	No Keratitis
With Corneal Vascularisation	0	0	1 (*a*)
Without Corneal Vascularisation	3	40	1 (*b*)

"Corneal vascularisation" here means the bilateral type found in ariboflavinosis (with or without the localised type found in rosacea).

Number of Cases Treated	Treatment	Response
10	Riboflavine only	Nil
10 (the same patients as above subsequently treated with——	Riboflavine and routine treatment	Healed
23	Riboflavine and routine treatment	Healed
10	Routine treatment without riboflavine	Healed
1 (*a*)	Riboflavine only	Healed
1 (*b*)	Untreated	Nil

One case (No. 1 (*a*)), with slight cutaneous rosacea and with the corneal vascularisation typical of ariboflavinosis but without keratitis, was treated with riboflavine alone and within a month the corneal vessels were visible only as " ghosts " ; the cutaneous condition remained unchanged. This was the only case in which gastric hypochlorhydria was improbable but riboflavine deficiency likely to occur, as the patient had slight symptoms of duodenal ulcer. It was the only case of the series which responded to treatment with riboflavine alone.

In the group of twenty-three cases, two required peritomy and one failed to heal until tonsillectomy was performed.

Seven of the series were examined by Sydenstricker, who confirmed ariboflavinosis only in case 1 (*a*), which was not a case of rosacea keratitis, and was the only one to respond to riboflavine therapy.

Conclusion

It is to be noted that, in the present series of forty-five cases of rosacea, there was only one which had the clinical signs and a history suggestive of riboflavine deficiency, whilst all had cutaneous rosacea; whereas in the series of thirty-six cases published by Johnson and Eckardt, thirty-four gave histories suggestive of a deficiency of this vitamin in their diet, and only nine " showed some cutaneous type of rosacea."

In the more recent contribution by Johnson[4] the description of the corneal lesions is typical of rosacea keratitis even in the absence of confirmatory cutaneous lesions, but the type of corneal vascularisation diagnostic of ariboflavinosis is nowhere mentioned by Johnson, nor does he state the condition of the unaffected eye in unilateral cases of " so called " rosacea keratitis.

It has recently been established[5] that the concentration of vitamin B2 in the corneal epithelium of the ox is very high as compared with other parts of the eye, and it may be that in patients with corneal lesions who are already generally deficient in this vitamin owing to faulty diet, or who may be locally deficient in it owing to injury to the corneal epithelium, riboflavine therapy promotes healing of the cornea; but in this present series of forty-five cases, rosacea keratitis does not appear to be the direct result of ariboflavinosis.

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2. ——— *Arch. of Ophthal.*, November, 1940, Vol. XXIV.
3. SYDENSTRICKER, SEBRELL, CLECKLEY and KRUSE.—*Jl. Amer. Med. Assoc.*, June, 1940, Vol. CXIV.
4. JOHNSON.—*Amer. Jl. of Ophthal.*, November, 1941, Vol. XXIV.
5. PIRRIE (pending publication).—The figure obtained for corneal epithelium is 2 γ per gramme wet weight; this is much higher than in the surrounding ocular tissue including the aqueous.

THE PROBLEM OF THE HYPERMETROPIC MINER

BY

A. CHRISTIE REID

NOTTINGHAM

ALL familiar with working conditions in a coal mine are aware that the myopic miner who consistently wears his correction on the top can dispense with his glasses below ground, and can, even with a considerable error, work without these up to an age that is often surprising, even at the coal face under modern strenuous conditions. Not so the high hypermetrope who has to accommodate to see his work, because a miner's work is done mostly within arms' length.

I am not aware that any systematic enquiry into the working capacity of the latter has ever been made, and the remarks in this paper may serve as an introduction to the subject.

In the last two or three years I have collected some twenty cases of hypermetropic miners, who have come to me or been sent for compensation reports, in whom the error had to be taken into account in assessing the working capacity. Most were complicated cases in which other factors were at work, *e.g.*, amblyopia in one eye, loss of one eye, corneal nebulae, nystagmus old or recent; and it is often a very difficult task to estimate fairly the emphasis one should put on the refractive error apart from these.

No compensation would be allowed for an error alone, but if an accident had spoiled the better eye, say, in a squinter, the visual result might be so poor that the man would have to leave the pit, being thrown back on to an eye with very poor vision. One asks, therefore, whether any standard of manifest error might be accepted as barring a man at a specific age from working in the pit, and also what symptoms, if any, may arise from the strain involved in the more or less continuous act of accommodation required for accurate work in the dim light of the mine with dark-adapted eyes.

All cases to be discussed save the last one had a manifest error of over two dioptres in the best axis of the better eye. One would not expect to meet with any serious difficulty below fifty years of age with less than this.

The general nervous make-up of the man must be taken into account, and one does meet with a few cases in which even a moderate error seems to lead to a train of nervous symptoms that demand a change from the pit bottom to the surface at a comparatively early age. I have three cases that have had to leave

the pit before thirty in whom, with both eyes not far from normal and with no appreciable nystagmus, symptoms were fairly severe.

CASE 1.—C.S., aged 28½ years, had come out only fourteen days before seeing me. He had never had glasses. Complaint: terrible headaches over months while doing face work. Unaided vision R. 6/36 one letter, L. 6/18. R. with +3·5 6/9. L. with +2·5 6/9. Together 6/6. As he said he could not go on he was advised to leave the pit, and agreed to do so.

Perhaps it should be explained that the most strenuous and the best-paid work is at the face getting the coal, or "ripping work" at the face, usually involving a good deal of pick work, even when cutting machinery is used, and in poor light. The pace is severer since the working hours were shortened, and the physical strain, apart from eyes, very great. Maintenance work on the roads up to the face is considered a degree less exacting.

CASE 2.—W.H., aged 30 years, wore glasses at school, gave them up, but at 22 had to resume for wear out of the pit. R.V. +4 with +0·5 cyl. 180° 6/12 slowly. L. +5 with +1 cyl. 165° 6/36. He had done ten years at the face but for the last eight months had not made a full week because of headaches and dizziness. Had to cease work a month ago. Certifying surgeon found no nystagmus. Referred to me. Unaided V. R. 6/60. L. less than 6/60. Glasses correct. No nystagmus. Advised to work on surface in glasses.

CASE 3.—R.R., aged 27 years, was at coal face without serious difficulty till sixteen months ago, when he received a severe blow at R. inner orbital angle. The eye not affected. This may have upset his nerve, for though he tried the face again, after a year of irregular work he had to give in on account of severe headaches. He then took to dairy work with great relief. He had no glasses before the accident. I found unaided V. R. and L. less than 6/60. No nystagmus. R. with +4·5 and +1·5 cyl. 150° 6/9 part. L. +5 and +1 cyl. 140° 6/24. I gave opinion that he never should have gone down the pit and was not entitled to compensation.

I am afraid that cases such as these three are too common. Many are wrongly certified as nystagmus, which may be present, but it may not be of a degree to incapacitate. Once branded as a nystagmic, the miner has difficulty in getting employment of any kind elsewhere, while if he remains at his own pit his case is a prolonged source of difficulty to the management.

How prevent such from going down? Advice to parents before leaving school does some good, but a few fairly high hypermetropes are not spotted at school if they have no squint, and

especially if both eyes function well. Every case of squint applying for pit work should be referred to an ophthalmic surgeon. A few alternators may be passed by him if the error is not high, but any with a definite amblyopia without correction should be rejected. Routine pit-head vision testing is impractical as being liable to be referred to a non-expert. Witness a recent case I had to get out of the pit—a lad of nineteen who had adopted the old dodge of crossing his hands and blocking the amblyopic eye while being tested. It is a kindness to bring such cases of early breakdown out of the pit while not too late for them to learn another trade.

This leads to the consideration of a second group, of which I can quote eight examples in which an amblyopic eye with or without a squint is present. In these there is perhaps more liability to accident—hence, more come to examination. These cases had to give up work at ages from twenty-eight to forty-two, but they need not all be detailed.

CASE 4.—Seen at clinic, aged 30 years, found to have +3 error manifest. R. corrected 6/9. L. less than 6/60 and no central fixation. No serious accidents. Glasses given for wear out of the pit, and to persevere in the pit. This he did for five years, then developed nystagmus and left the pit. He now reports, at thirty-six years of age, after a period of work on the bank, great improvement in his nystagmus symptoms and can do full work on the top.

I do not regard the error of refraction as having contributed to any nystagmus that may have been present in any of my cases. This point has been gone into on many occasions since my article on Miners' Nystagmus in the British Journal of Neurology, *Brain*, 1906, in which I sought for a physiological explanation for nystagmus, and found it in a disturbance of the equilibration of the eyes. It is doubtful if we have gone much further than this since then. I have still to find a miner who wears plus glasses at face work. I have heard of a few who have worn minus. But all the men and all the officials I question scoff at the idea of continuous wear in a hot, dusty place such as most of the men have to put up with. I have never been able to persuade a man to try it.

CASE 5.—T.L., the oldest in this group, actually carried on in the pit despite a high error till 42½ years, then a severe blow spoilt the L. eye (which I think must have been a good eye) and made him come out. After the accident, R.V. +6·5 6/9. L.V. +7 less than 6/60. Wearing glasses, he is now doing labouring work at a munitions depot, but reports, five months after the accident, that his eyes become blurred towards the end

of the day, and that he has difficulty getting home in the black-out. One accepts this, knowing how loss of sight in one eye in a man over forty years makes him very helpless in dark-adaptation conditions.

CASE 6.—A.S., aged 30 years, is one of a kind that we are meeting with under war conditions—a man who left the pit under my advice in 1940 and did good work on the bank, but who went down again under pressure after three months. Though put to work below on roads only, he broke down after two years with severe headaches. This was despite wearing glasses below to correct a high error. R. +4·5 with +2·5 cyl. 90° less than 6/60. L. +5 with +1 cyl. 90° 6/9 part. This man was advised to come out finally. It was a big risk going down again, and he should not have agreed.

CASE 7.—W.P. illustrates the difficulty of getting a high hypermetrope off the compensation list. Seen at thirty-six, he had had compensation for five years following a blow on the R. eye. He had worked these five years on the bank. I found no sign of trauma, but a squinting amblyopic eye that could not fix. Error about +4 with +2 cyl. at 90° less than 6/60. L. +3 with +1 cyl. 90° 6/9. No nystagmus even after exercises. I agreed that he was not a case for compensation, and I doubted how long he had really been entitled.

CASE 8 is a border-line case with a degree of nystagmus. W.H.S., aged thirty-eight years, came out with nystagmus at thirty-one and, after surface work till thirty-four, went down again and stuck it till two months ago, when he came up for illness. Having recovered from this, the question was should he go down again? I found R. +2·5 6/12 slowly. L. +3·5 6/60. A very fine nystagmus was seen with the ophthalmoscope, but not aggravated by swinging body movements. There was no characteristic attitude and no photophobia. He did not notice lamps dancing when last down the pit. I advised him to resume work on the top wearing glasses, and that compensation should cease. I was of opinion that it would be asking for trouble on both sides to send him down again.

Other such border-line cases between the hypermetropic syndrome and that of nystagmus could be quoted. Some of them are accident liable, and even on road maintenance work good near vision is essential for the detecting of flaws in the roof, defective wooden props, etc. (Steel props do not bend but may break suddenly without warning.)

CASE 9 was fortunate in having a safe job on a road haulage stationary engine below ground. J.E.W., aged thirty-seven years (R. +3·5 with +0·75 cyl. 90° 6/9. L. +4·5 with +1 cyl.

75° 6/24), had had to give up general maintenance fitting work below as he "could not wear glasses at this work" because of dust and sweat. Despite his complaint of retro-ocular pain and a decided light sensitiveness during examination, I advised him to carry on with his engine. He had worn correcting glasses at this work.

Four of my cases had returned, in late middle life, for one reason or another, to the pit after a spell on the top, but could not stick it after a certain period.

CASE 10.—E.W., aged 59 years, after fifteen years surface work, went down again on the national emergency call for more coal getters. After two-and-a-half years with increasing discomfort, he had to give up owing to a series of minor accidents and severe headaches. R. +4·5 less than 6/60. L. +3·5 6/6 part. He had been wearing glasses on the top but could not at the face.

CASE 11 was in a sorry plight with "nerves," and had had several accidents. S.E., aged fifty-two years, got glasses, for near work only, some years ago. He had left the pit because of eyes nine years ago and had worked well on the top. On appeal, he went down again, but after eighteen months had to cease. No nystagmus. R. +3 with +1·5 cyl. 70° 6/18. L. +3·5 with +1 cyl. 90 6/9. Two months after coming out again he came to see me, looking a new man, and was doing full work on the top.

CASE 12.—W.B., aged 48 years, had been "ripping" (*i.e.*, on road maintenance work in the pit) for four years after losing the L. eye from an acute septic (hypopyon) ulcer. The error was not high. R. +1 with +2·25 cyl. 110° 6/9 part: unaided 6/18. He had this, but had not worn it in the pit. He had now daily headaches, felt uncertain at work, numerous minor accidents, off work last three weeks with bruised back. I had no doubt about his willingness and also his disability, and advised him to come out finally.

CASE 13.—J.S., aged 62 years, lost R. in accident at face. Has done light work on roads in pit for twelve years wearing correction +5·5 L. 6/18 almost: unaided less than 6/60; no nystagmus; eye otherwise normal. There is much dread of light, dizziness on stooping, and he is very helpless after dark. I reported that he was fit only for labouring work on the top, and considered that his symptoms were entirely due to eyestrain, but that this would have been manifest even if he had had both eyes, for in that case he would probably not have been given light work and any breakdown would obviously have been due to the large error alone. I considered that he had no claim for compensation now.

I shall quote only one other case, which is of interest as having been quite recently referred to a referee.

CASE 14.—J.B., aged 60 years, a highly intelligent pit "deputy" (supervisor), had no trouble till after a recent accident that led to loss of one eye. He had not worn distant correction before the accident even on the surface. The error was slight, +1·25 with +0·25 cyl. 90° 6/6: unaided 6/18 almost. Since the accident he had resumed work below (*i.e.*, going round the workings wearing the glasses), but headaches, severe and continuous, such as he had never before experienced made him feel unable to continue. I reported in his favour and advised that he should leave the pit, considering that had he had both eyes he would not have had the symptoms. This opinion was contested by another ophthalmic surgeon and the man sent to a referee, who upheld my opinion. Having myself an error of +1·5 sph., and only at sixty-five taken to wearing distance correction, I do not place much emphasis on an error of that grade in a man of sixty; but when such a man has suddenly lost one good eye, the discomfort and difficulty seem to be considerable in the dim light of a mine.

The above cases may be taken as fairly typical of the points I wish to raise. I find that of seven cases which had an error of between 2 and 4 dioptres in the lowest axis, the age of breakdown averaged thirty-seven years, the extremes being twenty-eight and forty-four years. Over +4 few can go on beyond thirty years at the coal face. With 6 or more probably no man should go down below, and few do. It would therefore seem reasonable to advise lads with both eyes good not to go down if the error is 3 or more, and squinters or amblyopes not at all. Much, too, can be done in advising parents when a boy is about to leave school. If a pit accepts a man with a defective eye, it is accepting a risk, for in addition to being accident-liable, accident to the good eye may render him incapable; and even accident to the poor eye in a hypermetrope may necessitate glasses much earlier than would have been the case otherwise.

Finally, one hopes that this enquiry may stimulate interest in the hypermetropes in other industries in which lighting conditions leave much to be desired. I know of none that puts so severe and continuous a strain on the worker. Perhaps night engine-drivers and airmen who fly at night approximate, but the work is not so prolonged. These already have been tested and selected.

As to the exact location of the strain, I do not think it can be all put down to the ciliary muscle. The mental interpretation of small visual objects may be a factor. We can call the symptoms a neurosis, but that does not make them less real. Is it possible that, in the conflict between the call for a wide pupil in the dark and that for contraction of the ciliary muscle, a conflict that is the opposite of what is demanded in daylight, we have the making of a neurosis?

GLAUCOMA*

BY

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THE term glaucoma is used by some as synonymous with a raised intra-ocular pressure, but it seems more correct to apply it to a definite pathological entity, a symptom complex in which raised tension is generally present. There are cases of glaucoma without increase of intra-ocular pressure, and I think that I am right in saying that there are hard eyes which are not strictly glaucomatous. We can classify glaucoma as primary and secondary, if indeed we are correct in designating cases of cyclitis with high tension as truly glaucomatous.

Primary glaucoma can again be sub-divided into two categories: *congestive* glaucoma, and *glaucoma simplex*. I regard these two conditions as completely separate diseases, having but one thing in common—a raised intra-ocular pressure.

Congestive glaucoma is like a strangulated hernia, and is due to the formation of a vicious cycle. It may attack an eye which is, as far as one can discover, perfectly normal. Thirty-five years ago I performed my first operation in England at a private house in Coventry. It was an immediate success. I saw the patient many years later. He had never had any further trouble with the eye, and I could find no sign of glaucoma. Although I hold that the two classes of glaucoma that I have mentioned have little if anything in common, yet a case of congestive glaucoma may pass into a chronic stage which is quite unlike glaucoma simplex, and glaucoma simplex can develop congestive signs, and pass into a stage similar to that of a chronic congestive case. There is at present no certain knowledge of the cause of congestive glaucoma, but it is probably due to a physico-chemical derangement which leads to absorption of water by the vitreous, which swells, pushes the lens forward, and so blocks the iridic angle. This closure of the exits leads to more congestion and further advance of the lens, till the aqueous circulation ceases and the vicious cycle is complete. It is obvious that whereas there is free communication between the retro-lental space and the posterior chamber, no increase of the intra-ocular pressure can of itself alter the position of the lens. This can be attained by three conditions: an accumulation of fluid in the supra-choroideal space; general swelling of the choroid from vascular engorgement; and swelling of the vitreous. There

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is no evidence that fluid accumulates outside the choroid, nor can I think that hyperaemia of the choroid can attain such proportion that it can thrust the lens forward. We are left with a swollen vitreous. This seems to me to be the most probable cause of the initial rise of intra-ocular pressure in congestive glaucoma. There is no relationship between the general blood pressure and the ocular tension.

The signs of congestive glaucoma are mainly those of congestion, which may be moderate or intense. The general appearance in a typical well-developed case is pathognomonic. The conjunctiva is engorged and there is ciliary injection. The cornea is hazy, the pupil semi-dilated and irregular. The anterior chamber is shallow, and the pupil area greyish rather than black. Examination of the cornea with lateral illumination and the loupe shows that it is oedematous and that in the general steamy surface there are clear lacunae. This haze of the cornea may be partly due to polarization of the light reflected from the corneal lamellae. The late George Young, who used to give the lectures in optics for this course, showed that if a pig's eye be squeezed the cornea at once becomes milky, and he suggested that the increased intra-ocular pressure made the corneal lamellae slide over each other and induce polarization. Often the fundus is invisible, but when the media are sufficiently clear "flashing" of the arteries may be seen. I avoid the usual term, "visible pulsation," for this gives an entirely wrong impression of the actual picture. The whole arterial system flashes like the beam of a French lighthouse. It is not at all like the visible pulsation of a retinal vein, which is quite a slow process and has no relationship to glaucoma, although Elliot held a contrary view. It is possible to make the retinal arteries flash in this way in a normal eye by pressure on the globe. If an eye is predisposed to an attack of congestive glaucoma this flashing may be induced by quite gentle pressure, and this is a most valuable diagnostic method. In a very acute case the iris vessels may be injected. The mesoblastic part of the iris consists mainly of thick-walled blood vessels, and some of these at any rate are capable of resuming their embryonic function of carrying blood.

There are often definite prodromal attacks of congestion which are characterised by pain, slight or severe; by obscurations and dimness of vision; often by vomiting; and by coloured haloes round lights. The attacks may become more and more severe till one day the vicious cycle closes and glaucoma is present. Congestive glaucoma is accompanied by severe, often agonizing, pain in the eye and brow, there is dimness of vision, sometimes blindness, and severe constitutional symptoms. There is a rise of body temperature, and often *severe vomiting*. This most

characteristic symptom is of supreme importance because these general symptoms lead to a diagnosis of "influenza," with accompanying ocular neuralgia. Much valuable time may be lost and treatment of the most detrimental kind may be applied to the eye. One well-known ophthalmologist of the past said that he would like to rent permanently a whole page of the *British Medical Journal* and on it print, in leaded type: "*Vomiting is a common symptom of congestive glaucoma.*" A child who develops a rise of ocular tension after a discission of the lens does not complain of pain—*he vomits*.

The diagnosis of congestive glaucoma is generally easy, but may be very difficult. The only disease that ought to be confused with it is irido-cyclitis with rise of tension. We ask ourselves: Is this a case of glaucoma or of irido-cyclitis? Am I to use atropine or eserine? There is a simple answer: You are not compelled to use either; just order normal saline irrigation, and perhaps diathermy, and wait and see. The diagnosis may be made by careful use of the loupe and of the slit-lamp. This may reveal keratic precipitates and an albuminous aqueous. One does not find "K.P." in a pure glaucoma, and I think that a highly albuminous aqueous is strong evidence of cyclitis. Some think that there is no real distinction and that many acute glaucomas are secondary to cyclitis. Be this as it may, whether or no the basic causes are identical, the treatment is widely different and a clinical diagnosis is imperative. But in some cases the cornea is so hazy that the slit-lamp is useless and the diagnosis may be impossible. In such cases, after a short period of expectant treatment, I would use eserine, and if this appears to be harmful, then try homatropine rather than atropine. If this treatment is obviously wrong, then the dilatation of the pupil caused by homatropine is more readily overcome by eserine than the action of atropine.

The old idea was that once glaucoma was diagnosed, an operation must be undertaken at once. Every hour was precious. This is not the case, and I think that to-day few surgeons rush to an immediate operation. Once, at The Warneford Hospital, some 25 or more years ago, I saw a case of violently acute glaucoma. Both eyes had been treated, I think, for a fortnight with atropine; both were stone-hard with widely dilated pupils, and perception of light was abolished—a truly desperate case. I trephined both eyes and the result was incredible. The eyes became soft and lost all inflammation, and eventually both recovered, with a final acuity of 6/12. This astonishing outcome cannot be taken as an example for it is most unlikely to happen again, but it shows that it is not necessary in the ordinary case to rush to operation upon a highly inflamed eye when all the conditions are unfavourable for an operation. However violent the glaucoma,

I would always try the effect of eserine instilled every two hours till the pupils contract, and use diathermy. Hot pads and the wooden spoon are relics of a past age, for we now know that however hot we make the lids, short of actual scalding none of the heat reaches the eye. This has been proved by placing a thermopile under the lids.

There is no reason for not trying diathermy, but if the patient does not like it I would not continue its use. I rather tend to regard diathermy as a passing fashion, and have seen little, if any, good follow its use in any ocular condition; but others are most enthusiastic and so I must not dogmatize. The patient is so very unhappy that I see no reason to add to his discomforts by free purging. This has come down to us from the middle ages, but there is not an atom of evidence that purgation has any action in lowering intra-ocular tension. Rest in bed and eserine is the proper treatment. Injections of hypertonic solutions into the veins might be useful, but I have not tried it. I have seen no good effect from the use of dextro or laevo-glaucosan.

If this expectant treatment results in the pupil becoming small and the eye getting white, and if the tonometer shows that the tension is normal, then I think that the correct procedure is to do nothing active. The glaucoma has been overcome. It may never return, but it probably will. Let the patient have a 1 per cent. solution of pilocarpine to use every night, with instructions to push it should he note any recurrence of his original symptoms (pain and misty vision), and at once obtain the advice of a specialist. He must remain under constant observation. If he is the sort of man who will forget his troubles and let things slide, or if he will be living in a place where he is far from any ophthalmic surgeon, then an operation must be considered, and this must be undertaken if the tension does not return to normal.

Having decided that an operation is necessary, we must consider the best method. It is usual to perform an iridectomy, and many still adhere to what is called "the classical iridectomy." This is a horrible mutilation in which two-fifths of the iris, or even more, is removed. In London the section used to be made with a cataract knife, and some still adopt this instrument. I have always used a keratome, and I think that this is the instrument chosen in most clinics of the world. The chief object of the operation is to clear the iridic angle; the mere size of the coloboma has no merit. In order to effect this with ease and safety I, many years ago, devised my "trap-door" operation. I have described this elsewhere,* but in the main it is a trephine in which the disc is not removed, but replaced. The operation begins as an ordinary trephine operation, except that it is not necessary to cut a wide

* *Brit. Jl. of Ophthal.*, December, 1932. *Trans. Ophthal. Soc. U.K.*, Vol. LVI, 1936.

conjunctival flap. The hole is cut so that the disc remains attached at the upper aspect as described by Elliot. The iris almost invariably prolapses and is excised. The disc is now replaced and the conjunctiva sutured in the usual way. There are many advantages in this operation and, as far as I know, no disadvantages. There is no danger of wounding the lens. There is no post-operative astigmatism which may be present if a cataract knife is used. In many cases there will be slight filtration, but rarely enough to cause an ectatic scar. I have come to the conclusion that generally, if eserine has failed to bring the glaucomatous attack to an end, then mere iridectomy will be insufficient to prevent recurrences. This is where the slight filtration that may follow the " trap-door " method is so valuable. I do not care for iridencleisis in acute cases, and if the anterior chamber is shallow it is almost impossible to perform it without danger to the lens capsule. I have seen cataract with a tear in the capsule follow the " trap-door " operation on two occasions. I think that the capsule becomes very friable in acute glaucoma, and the mere removal of the slight support of the iris may lead to a rupture. In neither of these cases had any instrument been introduced into the anterior chamber, and yet there was, in one case, a triangular tear in the capsule just as though it had been wounded by a keratome.

If an operation is unsuccessful, then one must think of the presence of a tumour in the eye, or of thrombosis of the central retinal vein. If the other eye shows no sign of glaucoma, and if the vessels are healthy, then it is very probable that the glaucomatous eye contains a tumour. I have seen two cases of this nature recently. If operation fails to cure the glaucoma, remove the eye.

Recurrence is not uncommon after a simple iridectomy, and in this case it is wise to trephine the eye. If, however, the operation is successful, the cure may be permanent, and one does not see that slow deterioration that we shall have to describe in cases of glaucoma simplex.

In the absence of more certain knowledge, we can say only that glaucoma simplex is the result of senile changes in the eye and, I think, mainly affecting the optic nerve. Priestley Smith thought that it was caused by enlargement of the lens, and those who read his book (as all should) will almost be persuaded by the logic of his facts and the theories he deduces from them. But is there any real evidence that the lens is unduly large in the majority of eyes suffering from this disease? I am very sceptical whether there really is such a thing as a glaucoma caused by an intumescent lens, or whether the lens ever is intumescent. Does the lens, in fact, ever " swell "? Probably in an eye otherwise predisposed to glaucoma an unduly large lens may be the final act in that train

of events that end with a glaucoma simplex with raised tension. Close attachment of the iris root to the back of the cornea, the so-called *soudure de Knies*, is another suggested cause. In many cases of glaucoma the iridic angle has been found on section to be widely open, and the gonioscope shows the same condition. The *soudure* is an effect—not a cause. Increased production of lymph due to changes in the cervical sympathetic is a mere speculative cause. There is no evidence that arterio-sclerosis has any aetiological significance. We must content ourselves with the conclusion that we do not know the cause of glaucoma simplex, but we believe that, like cataract, it is a senile degeneration. In the majority of cases glaucoma simplex has no symptoms till it has been developing for a long time — perhaps for years. Many of our patients are not aware, till we tell them, that there is anything wrong with their eyes. Sometimes, indeed, we are the first to draw their attention to the fact that one eye is actually blind, or that they have a very contracted field of vision. Pain is unusual and is present only in some of those cases where the tension is raised or when congestive signs have been super-added to the simple state.

I wish to emphasise the fact that there are only three important and characteristic signs: changes in the optic disc; alterations in the fields of vision; and increase in the intra-ocular pressure. The last is the least important as a diagnostic sign, and may be absent.

Glaucomatous cupping of the optic disc when fully developed is so typical that it ought not to be mistaken for anything else. The cup comes close up to the edge of the disc; its edges are sharp, and are often undercut so that the path of the vessels is lost as they dip over the edge; the vessels reappear in a different plane and seem to have lost their continuity. But in its early stage the glaucoma cup may be difficult to diagnose. There is a shallow, sharp-edged, senile, saucer-like cup identical in appearance with the nascent glaucoma cup, and it may show the same pallor. It is never as white as the advanced glaucoma cup, which often shows what the French call a *nacré* hue—in plain English, mother-of-pearl. In some cases of tabetic atrophy there is a definite cup. Once I was completely deceived and spent a long time trying in vain to obtain confirmatory signs of glaucoma.

Finally, there is a congenital deformity which copies faithfully a fully developed glaucoma cup. In my young days I once confidently diagnosed a glaucoma cup in a young male private patient, but my view was negatived by an older and more experienced consultant. I lost *kudos* but gained knowledge. I saw the man many years later. There was no alteration in the appearance of the cups, and no other signs of glaucoma. If you will make a practice of examining with the ophthalmoscope

every patient that you see in clinic or hospital you will occasionally pick up this kind of oddity. In a school clinic it is easy to have a peep by the indirect method—one rather neglected in this country. May I remind you that if you are emmetropic or hypermetropic you will not be able to get a clear fundus picture unless you put up a +2·0 D. sphere behind your concave mirror. The indirect method is of great value in assessing the comparative tint of the two discs; it will show up a small difference in pallor that is not so easily recognised by the direct method. Again, the colour value of the observer's two eyes is not always the same, so if you are in doubt, examine both of the patient's eyes with one of your own. Again, the illumination of the instrument has much to do with the colour of the disc. If you have just had a new battery you will be apt to see pale discs. All these factors must be taken into account in estimating the colour of a glaucomatous disc. To become proficient, never miss any chance of examining normal fundi. There is a very wide margin between the normal and the abnormal, and this is important when you have to decide whether papilloedema is present or absent.

What is the cause of cupping? I am sure that most of you will say that there is an obvious answer. It is, of course, due to the raised intra-ocular pressure driving back the lamina cribrosa. This view was taken in the pre-tonometric era, and is, I imagine, still current. Is it true? I think that I can advance evidence that will at any rate make you begin to wonder, to think and, I hope, to investigate. Some years ago a man came to the Coventry Hospital who had been struck in the eye by a fragment of steel that had flown from a fractured drill. An X-ray picture happened to show the optic nerve quite clearly, and it was seen to contain the large mass of metal that I am handing round for your inspection. When the eye had been excised I tried to remove the steel with artery forceps—in vain. I had to get a pair of pliers to extract it. This is the structure that has to be cupped by the intra-ocular pressure, and yet the eye does not bulge at the intercalary region where it is most vulnerable. Then I find that cupping is not present in cases of congestive glaucoma when the eyes are stone-hard, even if this enormous pressure has been acting upon the optic nerve for quite a long time. On the other hand, I find deep 100 per cent. cups in eyes that have never shown any rise of tension as determined with the tonometer. But I am told this is true if the tension is estimated in the day-time, but if you try at night you will find hyper-tension. This argument seems to me to be mere casuistry. Schnabel, some thirty-five years ago, published an article pointing out that eyes which he had operated upon for glaucoma simplex might, during the few weeks that

they were in the ward after the operation, and with a now normal tension, develop a glaucoma cup. I have myself seen this happen more than once. In one case at the Coventry Hospital I made a note, "No cup." A month or so later I noted, "Shallow cup," and after a few more weeks, "Deep cup." This case had only a moderate rise of tension—I think about 30 Schiötz units. I am unable to give you the exact dates and figures because the Coventry Hospital has been practically destroyed by enemy action, and I doubt whether the records could be found. Schnabel described a condition which he called *cavernous atrophy*, in which atrophy of the glial tissue in the nerve head causes large spaces to develop, causing a cup.

Pickard, of Exeter, has made a careful study of glaucoma without rise of tension, and has written several papers on the subject. I have seen many of these cases and have followed up a few of them for years. Some of them have shown no deterioration of function, but in others there is a slow, but unfortunately sure, downward course. Professor Enroth, of Helsinfors, told me, in a private communication, that in his experience the majority of cases of glaucoma simplex ended in total blindness whatever treatment was adopted. This has not been my own experience. I have seen many cases fifteen and twenty years after a successful operation who have maintained their visual functions unaltered. On the other hand I have not infrequently been asked to examine patients for blind pensions upon whom I had operated apparently with success in the past, and many of them have been blind. My own view is that the glaucoma cup is caused by a senile degenerative process in the nerve head which softens it and allows it to become cupped by very slightly raised tension, or even by the normal intra-ocular pressure. In secondary glaucoma deep cups are often present, so we must assume that the inflammatory process has the same action upon the nerve tissues that we have assumed in glaucoma simplex. In some cases the depth of the cup appears to diminish after operation, but in others, in spite of a normal or even a sub-normal tension, the process goes steadily on; the cup becomes total and the fields small, till eventually complete blindness results.

The alterations in the fields of vision that are seen in glaucoma may affect either the peripheral or the central part of the field, or both. The typical peripheral change is the formation of a nasal step. This quadrantal notch deepens till it approaches the fixation spot. Here it tends to halt for a time and the patient alternately sees and loses the object he looks at. But one day the step reaches the centre and then central vision is lost. In my own opinion no time should be lost in a case of this kind; an immediate operation is called for. It has been taught that it is dangerous to operate—

that the intervention may just push the eye over into central blindness. My colleague at the Birmingham Eye Hospital, Mr. Charles Rudd, has just told me that he has recently experienced this misfortune with one of his patients, and there is no doubt that it occasionally happens. On the other hand, if nothing is done the end is certain, and generally the operation is successful. I have operated upon cases of this kind and have never seen immediate blindness follow. I had one case of this kind at Leamington—a private patient. She complained that objects kept going and coming, and she had the typical step impinging upon the central fixation spot. I performed a trephing operation, and followed up the case till the lady died. There was no further deterioration, and to the end she kept losing her central fixation and finding it again. In many cases the end stage of the field is a small central area all, say, within the five degree circle. These are not so dangerous as the deep steps.

The changes in the central part of the field are most important, and an examination with the tangent screen, the Bjerrum screen, must never be omitted, using small test discs and working at a distance of two metres. The first step should be to pass the test object along a circular path, about half way between the blind spot and the fixation spot, using the fixation spot as a centre. If there are any horn-like prolongations of the blind spot this path will intercept them. The common scotoma in this region is the Siedl. It consists of a horn-like extension of the blind spot along a course roughly concentric with the meridians on the chart. These horns may be above or below the central raphe, or in both situations. They may extend to embrace the fixation spot and implicate central vision.* I have seen this happen when the peripheral field showed no contraction. We may find peripheral contraction, central scotomata, or both. These central scotomata are not very common, but when present their effect may be devastating and quite unexpected—in fact, loss of central vision from this cause may be the first symptom of glaucoma simplex. Never omit the Bjerrum test. It takes a very short time to demonstrate the absence of a Siedl scotoma, but a long time to map it out properly. You will find full details of all these field changes in Traquair's Book on Perimetry.

Rise of intra-ocular pressure is, from a diagnostic standpoint, the least important of the signs of glaucoma simplex. In general it is not high, averaging from 30 to 35 Schiötz units on the old scale—say at most 40 units. Often it is much lower, and, as we have seen, may not be present. In the past there was much muddled thinking. Congestive glaucoma and its variants had not been clearly differentiated from the simple variety, and the tonometer was not in general use. I have made a most careful

study of this instrument from the time it was introduced by Schiötz thirty-five years ago. I tried the Sydney Stephenson instrument and found that it was unreliable. But I have not the slightest doubt that the Schiötz model is a thoroughly reliable instrument. This is, I think, the opinion held outside the British Commonwealth of Nations, but there are still some in the country who think that they can form a better judgment with their fingers. One still hears of the *tactus eruditus*, a term that used to impress medical students (special fingers given to special men). My own fingers are most untrustworthy. Without the tonometer I should have operated upon eyes not glaucomatous, and eyes calling for operation would have been left alone. At two meetings of the Midland Ophthalmological Society I asked the members for opinions on the tension of an eye and I got the most extraordinary answers: "Normal," "A little up," "Softish," "Hard." The tonometer at once gave the true reading. And yet I have been told by my colleagues that the tonometer is quite unreliable, and that "educated fingers" are far better.

Sometimes we get a case of glaucoma simplex with a very high tension. This is often a long-standing neglected case with a deep cup, small field, with shallow anterior chamber, and other signs indicating that a simple case has become complicated by congestive features. These cases call for immediate operation, probably more than a mere iridectomy, and they generally do badly.

Apart from the three signs that I have mentioned of glaucoma simplex, there are no others of any importance. It is said that changes take place in refraction and accommodation. I have never noted such. In many old persons the anterior chamber tends to get smaller, probably because the lens is larger than normal. The same may be found in glaucoma simplex, but a shallow anterior chamber is not a typical sign of the simple variety. These vague signs are those rather of senility than of disease.

The differential diagnosis is generally not difficult. I have already mentioned some of the conditions which may lead to difficulty. Certain cases of chiasmal lesion cause fields very similar to those of glaucoma, but they tend to be bilateral, and only a deep senile cup could lead to confusion with those forms of glaucoma which show no rise of tension. In all doubtful cases an X-ray picture of the sella turcica will make the diagnosis clear. It should be a universal rule to photograph the skull in any obscure change in the optic disc and fields. A slowly growing cataract may mask a glaucoma simplex, especially if bilateral, and no doubt mistakes have been and will be made; but the routine use of the tonometer will obviate many of them. The modern tendency to quick refraction, caused by the unsatisfactory staffing of our hospitals, by

the N.O.T.B., by rapid examination of recruits, and by the difficulties of the present time, are liable to lead to early cases of glaucoma simplex being overlooked, and this is more likely to occur to those who carry out refraction without a general knowledge of ophthalmology.

The most important question that we have to decide is whether we ought to operate, and when. I think that one may say that as soon as it has become obvious that, in spite of treatment with meiotics, there is a definite deterioration in function, then an operation is necessary. The age and general physical condition of the patient must be considered.

Many procedures have been employed to cure glaucoma simplex. I have tried iridectomy, Herbert's small flap operations, and punch operations. These have not been satisfactory in my hands. Trephining has so many disadvantages that I reserve it for special cases. The drawbacks are: the frequence of an ectatic scar that is unsightly, often uncomfortable, and apt to be a channel of destructive late infections; and late formation of the anterior chamber, with consequent development of a nuclear cataract, a serious and not very uncommon event. Apart from this form of cataract, my own statistics show that 10 per cent. of all trephine operations are followed by cataract, and this figure agrees with that published by others. For the past fifteen years or more I have chosen iridencleisis as my usual operation. I have now done about one hundred-and-fifty cases and I am satisfied that as far as I am concerned it is superior to trephining. The operation is not easy, but the results are very good and there are few complications. Cataract is a rare sequel and late infection has not been observed. I have seen many cases up to fifteen years after operation with eyes still functioning normally. Only this month I have examined two. One man, aged seventy-one years, had an iridencleisis ten years ago. He has only one functioning eye, but this has an acuity 6/12. There is a shallow pale glaucoma cup and the field is very small. But he does good work for the country, making fine tools in a munitions factory. He has no cataract. The other eye is blind. There had been two trephining operations and the lens is opaque. In the other case a trephine operation had been done on one eye. There is a nuclear cataract in this eye. The other had an iridencleisis at the same time. The lens is clear and the vision good. I have recently had one case of sympathetic ophthalmitis in a very decrepid old man. He never came up after the operation till the inflammation was advanced and past treatment. Of course, similar conditions have followed trephining and any other operation upon the globe. I have had no experience of cyclodialysis. I used to see many cases of Herbert's wedge isolation performed by a colleague, but I was not impressed with the results.

Time does not allow me to deal with secondary glaucoma, with juvenile glaucoma, or with buphthalmos.

Many of the views that I have expressed in this lecture are heterodox. I hope that you will regard them as my own opinions, but that you will keep them in mind and try to ascertain in your own future practices to what extent they are true or false.

SOME REMARKS ON FINCHAM'S CAPSULAR THEORY OF ACCOMMODATION

BY

J. W. NORDENSON

STOCKHOLM

SOME years ago a new theory of accommodation, called the capsular theory, was presented by Fincham.* It was an attempt to conciliate the two diverging theories of v. Helmholtz and Tscherning. Fincham agreed with v. Helmholtz so far as to accept the idea that the alteration of the curvature of the lens was due to a slackening of the zonula, allowing the elasticity of the lens-capsule to exert its influence; on the other hand he accepted the idea of Tscherning that the lens in accommodation takes a conoidal shape, forming a lenticonus anterior. According to him this conoidal form is due to the fact that the capsule of the lens is of varying thickness in different parts. As this capsular theory, which has attracted a certain amount of interest, is incompatible with certain facts about accommodation, I here allow myself to advance some critical remarks on it.

In forming an opinion on this new theory we have to consider two questions. The first is whether the lens really assumes a conoidal form in accommodation; the second is whether a conoidal form can be caused by the varying thickness of the lens-capsule in different parts.

Before entering on the first of these questions—whether the lens assumes a conoidal shape or not—it is necessary to remark that Fincham's conception of a conoid does not coincide with the generally accepted scientific definition of this word. Thus the ancient mathematicians used it to denote a surface, engendered by an element of a parabola or a hyperbola rotating round its axis, in other words a truncated rotation—paraboloid or hyperboloid. In later times it has been used to denote the surface engendered by a straight line—generatrix—running both along a

*FINCHAM, E.—The Mechanism of Accommodation. Monograph Supplement VIII. *Brit. Jl. of Ophthal.*, 1937.

curve—directrix—and along a straight line—axis—being constantly parallel to a given plane—planum dirigens. Fincham's conception of a conoid on the other hand does not seem to coincide with any of these definitions; he obviously holds that it is a segment of a sphere, resting on a truncated cone or, at any rate, on a body of rapidly decreasing meridional curvature. The following argumentation is based on the assumption that this is Fincham's definition of the word.

Resuming the question whether the lens assumes a conoid form or not in accommodation, we now have to examine the arguments given in favour of a positive answer. The main argument is drawn from an experiment, which was devised by Tscherning in his time, and has now been repeated by Fincham. It was carried out in the following way: Four sources of light were arranged on an arc in such a way that the image of them, formed by the centre of the anterior surface of the lens, was a straight vertical line. The pupil was dilated, and the eye was accommodated about 5 dioptres. The fixation was first adjusted so that the line of images was formed by the central area of the lens surface; then the fixation was altered slightly, so that the images were formed away from the centre. In the latter case the two peripheral images were seen to be elongated and splayed out. This, according to Fincham, indicates "the shallow conoidal form of the surface."

This way of concluding, however, leaves room for several objections. No notice is taken of the influence of the forelying cornea, and no comparison is made with the conditions in the non-accommodating eye. The conclusions must therefore be very uncertain. The only permissible conclusion to be drawn from the experiment in question is that the curvature of the peripheral part of the apparent anterior surface of the lens is slighter than the curvature of the centre. It does not allow of the conclusion that the difference of curvature between centre and periphery is so great that the denotation "conoidal" is justified. (What is to be understood by "shallow conoidal" remains to be explained). The effect seen can easily be produced by an ordinary surface of the second degree.

If any conclusions are to be drawn about the form of the anterior surface of the lens from the images that it gives, it is necessary first to determine the influence of the cornea on the rays that reach the lens through it and are reflected. As I have had the opportunity of demonstrating in a previous investigation,* a study of the peripheral parts of the anterior surface of the lens,

* NORDENSON, J. W.—Uber die Form der Linsenflächen bei der Ackommodation. Skand. *Arch. f. Physiol.*, 1916.

preceded by a survey of the cornea, gives an aspect of the form of this surface in accommodation totally different from the one described by Fincham. The investigation in question consisted of a trigonometrical survey of the anterior surface of the lens, first in accommodative rest and then in accommodation, preceded by a similar survey of the corneal system, *i.e.*, of the anterior surface of the cornea. In that way due allowance was made for the influence of this system on the rays, that pass through it twice. Contrary to the assertions of Tscherning and Fincham the measurings showed that the radius of the anterior surface of the lens was diminished in accommodation, not only, as is generally admitted, in the central part, but also in the periphery. Thus the radius of the central element of the horizontal meridian temporal to the axis, was, in rest, 9°,54 mm. in accommodation 7°,38 mm.; and the most peripheral of the measured elements, situated about 20° temporally, showed a radius of 16°,2 mm. in rest and of 10°,62 in accommodation. Similar results were found on the nasal side; measurements in other directions were not made, the possibilities of obtaining exact results there being considerably less than in the said meridian. Thus these measurements showed that a very marked increase of the curvature of the anterior surface of the lens takes place, and that the radius of the peripheral parts, although still greater than that of the central parts, is, however, always smaller in accommodation than in accommodative rest.

As the measurements now quoted are somewhat complicated and difficult to undertake and cannot very easily be repeated by those wishing to form an independent opinion on these questions I shall here describe a simple method of controlling them: The eye of a young person, possessing a good range of accommodation, is dilated with cocaine. The person is then seated at a table with a chin and forehead support. At about one foot's distance from the eye a telescope of about three times magnification is placed. Vertically above the objective of the telescope is placed a strong slit-lamp. This slit-lamp is lit, and adjusted so that its image on the anterior surface of the lens appears in the pupil. A fixation mark consisting of a small mirror, mounted on a shaft, is then placed so that the images of the slit-lamp in the anterior corneal and in the anterior lens surface lie in a vertical line. The fixation-mark is hereby placed as close to the eye as possible, the patient still being able to read a small letter scratched on the glass of the mirror. The patient is then asked to fix alternately the letter and the image of a distant lumisource of light, which lies perspectively behind the letter, thus alternately accommodating and resting the accommodation without changing the direction of the visual line. If now the slit-lamp has been adjusted in vertical

direction in such a way that its image in the anterior lens surface in accommodative rest lies close to the pupillary border, one can see how in accommodation, it moves towards the centre. This decidedly contradicts the assumption of a diminishing of the curvature in these peripheral parts of the surface, which would take place, if a lenticonus were to be formed.

The first of the above questions—whether the lens in accommodation assumes a conoidal form or not—being thus answered negatively it might seem unnecessary to discuss the factors that, according to Fincham, would bring about this form. However, some remarks on this presumed mechanism may be allowed here.

Fincham is of the opinion that a conoidal form of the anterior surface of the lens in accommodation is brought about by the varying thickness of the capsule in different parts. According to him, the capsule in its anterior surface is about 1½ times thicker 2 mm. from the centre and according to the general theory of elastic spheres this difference should be sufficient to cause different curvatures there and in the centre. Experiments, demonstrating this fact, have been undertaken by Fincham, and they can easily be repeated by anybody by means of an ordinary football bladder, which generally has four seams twice as thick as the rest of the bladder, and which, when pumped up, shows a distinct bulging-out of the parts between the seams.

If the supposition that the capsule of the lens, owing to its varying thickness in different parts, should react like the india-rubber bladder when its tension is increased, were correct, the capsule would—as Odqvist* has pointed out—show a bulging-out of its thinner parts in the so-called Bowman experiment. The said experiment consists in placing the extracted lens in a hypotonic solution, in which it absorbs water and swells, the capsule being thereby considerably stretched. On inspection, the lens then presents a spheroid form and shows no obvious bulging-out of any part of the capsule. In order to ascertain, however, that this is actually the case, I have undertaken to measure the curvature of a meridional section of the anterior surface of the swollen lens.

The extracted lens that was to be measured, was placed in front of the Helmholtz ophthalmometer on a small tray at a distance of about 80 cm. Above it was placed a plane mirror that projected an image of the lens along the axis of the ophthalmometer, so that the measuring could be made in a horizontal position of the ophthalmometer. A small lamp was fixed close above the entrance to the plate-house of the instrument, and another was placed to the side of the ophthalmometer successively at a varying

* ODQVIST, B.—Studien ueber den Ackommodations Mechanismus. *Acta Soc. Med. Suec.*, 1937.

distance from the axis. The interval between the images of these two light-sources was then measured with the ophthalmometer, and from these measurements a trigonometric curve of a meridional section of the anterior surface of the lens was obtained. In a lens of a 55-year-old woman, extracted in the capsule, and placed in distilled water for a couple of days, the following results were obtained:—

Angular distance of element from axis	Approximative linear distance of element from axis	Radius after 4 hours	Radius after 48 hours
5°	0·5 mm.	6·3 mm.	8·1 mm.
15°	2·2 mm.	7·2 mm.	9·0 mm.
25°	3·8 mm.	7·4 mm.	9·2 mm.

Measurements of other lenses gave analogous results.

These measurements show that the lens actually assumes an almost spherical form when swelling, and that no bulging-out of the thinner parts takes place. The radius of the peripheral parts differs very little from that of the more central ones, whereas, if a conoid were formed, the radius would be of infinite magnitude. Likewise it would be considerably greater than in the centre, if the lens capsule, when swelling, assumed the form of a surface of the second degree.

As to the cause of the fact that the lens-capsule, when stretched, does not present the same reaction as the india-rubber bladder, Odqvist points out that is probably due to the fact that the capsule is not of a homogeneous structure and that therefore the general laws of distension are not applicable to this tissue.

ANNOTATION

Paralysis of the Abducens Nerve following Spinal Anaesthesia*

Hagman and Wood, of New York, report two cases of sixth cranial nerve palsy following spinal anaesthesia. They state that the abducens is affected in well over 90 per cent. of all cranial paralyses following lumbar anaesthesia.

*Annals of Surgery, May, 1942.

Some authors claim that the incidence of abducens paralysis is as high as 1 per cent. of all spinal anaesthetics; others speak of it as occurring once in 250 cases. During the past decade fewer cases have been reported and the authors postulate three possible reasons: (*a*) improvement in technique; (*b*) these cases still occur but are not recognized; (*c*) these cases still occur, are recognized and are not reported.

The aetiology of the paralysis is still not clearly understood, but the authors believe that "the problem will be solved on the basis of a disturbance in the dynamics of the spinal fluid during spinal anaesthesia."

Each of these two patients complained of headaches from the 3rd to the 6th day; diplopia was noticed on the 5th day in one case and on the 6th in the other. Recovery was slow but apparently complete at the end of a couple of months. It would seem to be a little queer that patients with sudden diplopia of this sort do not recognise it and the only case which persists in our own recollection bearing on the point, not it is true the sequel of spinal anaesthesia, was one which was referred to the writer by the house surgeon of the Nose and Throat department for an opinion on the external rectus paralysis present. In this case the eye was blind. The patient had not noticed any double vision but was quite positive that the eye was blind, as indeed it was.

ABSTRACTS

I.—MISCELLANEOUS

(1) **Rycroft, B. W., Major, R.A.M.C.—Night vision in the Army.** *Brit. Med. Jl.*, November 14, 1942.

(1) **Rycroft** presents an analytical and statistical report of night vision tests in the Army. "In any night vision test the object is to stimulate the parafoveal and peripheral areas of the fundus. The test object used is a large V mounted in five different positions on a translucent screen behind which is a small bulb run from a dry battery and controlled by rheostat and voltmeter. The lamp is at varying distances from each of the five screens, which are arranged as an irregular pentagon. The apparatus is rotatable so as to bring one screen at a time in front of the examinee."

Preliminary details as to age, existence of any eye trouble and the wearing of glasses, how is the sight in the dark, home, whether in town or country, work in civil life and that in the Army are recorded and the soldier sits at one metre from the apparatus and is rendered

fully dark-adapted by wearing dark goggles for half-an-hour. At the end of this period the goggles are cleared of moisture (a most essential procedure) and he then writes down the position of the five V's in turn. If he sees all five his night vision is assessed as 5/5 which is a very good result; 4/5, 3/5, 2/5, and 1/5 are average, and 0/5 is poor. When any unit has been examined the results are classified in 3 main groups. Men whose night vision is above average; average night vision; and those below average.

It has been found that age has a marked effect on the results; most men over 30 years of age are slow in the rate of dark adaptation and defective in night vision. Defective day vision whether due to refraction errors, myopia, corneal nebulae and familial defects influences night vision. Intelligence plays a part; the labourer often failing because he does not grasp the significance of the test. A proportion of cases occurs in which no explanation can be given; it may be due to steamy goggles or to malingering. The complete unit report which gives the facts, the assessment and other remarks is sent to the officer commanding. Results must be viewed in their proper perspective, with a due sense of appreciation of a man's general training and efficiency. In an infantry brigade 4 regiments were examined. The over-average percentage was strikingly constant in each, round about 31 per cent. A table is also given of percentage analysis of night vision defectives in 4 regiments. The author and his colleagues are to be congratulated on a valuable piece of work which makes for increased efficiency in night operations.

R. R. J.

(2) **Smith, F. G. W.—Observations on intra-cranial pressure during general anaesthesia.** *Brit. Jl. of Anaesthesia*, Vol. XVII, p. 85, 1941.

(2) **Smith** draws attention to the work on the relationship between intra-cranial pressure and retinal vascular pressure and on the variations in intra-cranial pressure that have been observed during anaesthesia. His own observations concern 42 patients in the third stage of general anaesthesia and ten controls. He found that in patients under general anaesthesia intra-cranial pressure was increased by 6·7 mm. Hg, the intra-ocular pressure by 5·6 mm. Hg and the retinal diastolic arterial pressure by 15·5 mm. Hg. The greatest average rise of intra-cranial pressure was in those cases which had intra-tracheal anaesthesia; these patients had nitrous oxide and oxygen chiefly and little ether; hence, despite a more free airway, they tended to conform to what is considered usual in gas anaesthesia. A case which had ethyl chloride induction and open ether showed the highest retinal diastolic arterial pressure, and one, under gas and oxygen only, the highest intra-cranial pressure. Three cases induced by pentothal sodium showed all readings

definitely lower than the average; those premedicated by opium derivatives and atropine were mainly higher. The intra-ocular pressure rose to 43 mm. Hg in two cases under nitrous oxide, oxygen and ether, and to 36 mm. Hg in one case under ethyl chloride and ether.

He concludes that intra-cranial pressure during anaesthesia may be estimated, approximately, by anaesthetists in a simple manner in cases where they consider this advisable, *i.e.*, septic, dehydrated, cerebral or renal conditions. They should observe with an electric ophthalmoscope whether spontaneous retinal venous pulsation was present, or whether light pressure on the eyeball through the lower lid by the finger was required to produce this. The tension of the eyeball should then be estimated by finger pressure or by the sight and sense impression afforded by producing a slight dimple on the eyeball with a glass rod. (Letchworth's procedure).

Where the eyeball was obviously softer than normal, this test would be of no consequence unless comparatively severe pressure was required to produce venous pulsation. The intra-cranial pressure might be considered to be within normal limits when:—

1. Spontaneous retinal venous pulsation was present.
2. Intra-ocular pressure apparently normal.
3. Gentle pressure on the eyeball would not produce a venous pulse, if this was not present already.

ARNOLD SORSBY.

(3) **Bangerter, A. and Goldmann, H. (Berne).—Studies on the angle of the anterior chamber in primary glaucoma. Choice of operation based on such findings. (Kammerwinkelstudien biem primären Glaukom. Operationswahl nach dem Winkelbefund).** *Ophthalmologica*, Vol. CII, p. 321, December, 1941.

(3) Using the Goldmann contact glass for exploration of the angle of the anterior chamber, **Bangerter** and **Goldmann** report:

1. The findings in 40 patients with simple, and 39 with decompensated glaucoma. The angle was always found to be open in simple glaucoma; in 55 per cent. of the cases characteristic changes of the angle (pigmentation, entanglement) were observed. In decompensated glaucoma the angle was completely closed during an attack. After relieving pressure by means of medicaments (where this was successful) the angle was seen to open, but remained very narrow.

2. The findings after iridectomy or cyclodyalisis in primary glaucoma indicate that:—

(*a*) As the aim of iridectomy is the freeing of the corneo-scleral trabeculae, only basal iridectomy, which includes the iris root, achieves this. It is the operation of choice with decompensated glaucoma with obstruction of the angle during the increase of pressure.

(*b*) Cyclodialysis is successful only when the angle is not too narrow. It is the operation of choice in glaucoma simplex with open angle.

(*c*) With inflammatory glaucoma, in which iridectomy has not produced full normalisation of pressure, cyclodialysis at the site of the iridectomy is effective. In simple glaucoma with narrow angle, iridectomy in the coloboma region should be carried out prior to cyclodialysis in order to avoid post-operative closing.

ARNOLD SORSBY.

(4) **Molnar, E. J. (Toledo, Ohio).—Cloves, oil of cloves and eugenol: their medico-dental history.** Dental items of interest, July-October, 1942.

(4) **Molnar's** pamphlet runs to 30 pages, and is well documented. It traces the use of cloves from the earliest times to the present day and is well worth reading by any who are interested in the history of drugs. The use of cloves in medicine began in Byzantium, probably because it was at that date the great clearing house for spices, etc., brought from the East and partly because most of the physicians were trained in Alexandria. Cloves was used for sore and weak eyes by the early Arabic physicians, and was also recommended by Celsus for sore eyes. Avicenna's clove pill was famous in the middle ages for the treatment of foul breath. Coming from so distant a source it was naturally an expensive drug. But its chief use was in toothache as a local application, and in halitosis. By the middle of the nineteenth century the materia medica no longer listed oil of cloves as beneficial in eye cases. The author has an interesting section on the influence of spices in world history. When the abstractor was a medical student one of his teachers used to extol the use of the mist. caryoph. for three reasons: it might do good, it would do no harm and it was pleasant to take.

R.R.J.

II.—CORNEA

(1) **Wetzel, T. O. (Lansing, Michigan).—Dendritic keratitis.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 409, 1942.

(1) **Wetzel's** paper contains a very full account of the historical facts in the recognition of dendritic keratitis as a clinical entity; its aetiology; sex, age and seasonal incidence; its clinical features, diagnosis and treatment.

It is believed that the disease is due to a filter-passing virus. The toxin operating on the nerve endings in the superficial layers of the cornea is un-identified. The primary focus is probably in

the bulbo-spinal root. The resistance of the body is lowered, in many cases by some acute pre-existing disorder of the upper respiratory passages such as influenza and coryza, the severity of which bears no relation to that of the keratitis. The degree of loss of corneal sensation varies with the tendency to recurrence and healing rate.

In the literature there is a dispute as to the presence of vesicles in the early stages of the disease. Some authors deny the existence of such. The author comments that the sex incidence is males to females 3 to 1, but under 16 years of age the sex ratio is equal, and over 16 there is a male predominance of 5 to 2. The disease seems to be more prevalent in moderately cool and wet weather. In India Wright described an increased incidence in September rising to a peak in November and December, and Kirwan in the same country noted its prevalence during the rainy season and the monsoon.

In describing the treatment of dendritic keratitis the author discusses the value of quinine ointment, 2 per cent. or as drops, 1 to 2 per cent. The effects of infra-red and ultra-violet light are unconvincing. The importanee of dealing with focal sepsis is evident in some cases. Vitamins B_1 and A are mentioned as beneficial supplements to healing. Chemotherapy with sulphonamides is unconvincing at present. The author favours the local application of iodine either in alcoholic solution or as a syrup widely around the affected area of the cornea.

H. B. STALLARD.

III.—PATHOLOGY

(1) **Guibor, G. P. (Chicago).—Adrenal neuroblastoma with orbital metastases.** *Amer. Jl. Ophthal.*, Vol. XXV, p. 387, 1942.

(1) **Guibor** describes the clinical features and the post-mortem findings in a white male infant, aged 7 months, who died 4 months after the onset of a neoplasm in the left orbit. The primary growth, a neuroblastoma of the right supra-renal, was not evident during life.

Clinically the orbital neoplasm was diagnosed as a sarcoma. The blood count showed a reduction of polymorphs to 9 per cent. and a relative increase of lymphocytes to 86 per cent. in a white cell count of 12,700 which suggested chloroma to the author. Subsequent radiographic appearances showed a sun-ray opacity in the left temporal bone and the radiologist favoured haemangioma as the diagnosis.

Metastases occurred behind the peritoneum, in the mediastinum,

frontal and temporal bones, dura, tongue, orbit, palate and tonsils. The liver and right kidney which lay against the primary neoplasm were not involved, neither was the brain infiltrated from the dural deposits, nor the long bones affected.

The author comments on the rarity of this neoplasm. 291 cases have been reported in 76 years. The rapid nature of the growth and the concealment of its primary source are features of note.

H. B. STALLARD.

BOOK NOTICE

Annual Report and Statistics of the Government Ophthalmic Hospital, Madras, 1941. Government Press, Madras, 1942. Price, 8 annas.

This statistical report follows closely the lines of previous publications. The superintendent responsible is now Rao Bahadur K. Koman Nayar, whose name is familiar to our readers. A new feature is the account of work done in connexion with air raid precautions. The training school in ophthalmology has been maintained and besides training of a large number of medical students there has been a substantial post-graduate course. Training and protective measures in A.R.P. have also been undertaken. The usual tables of diseases treated and operations performed show that much work has been performed in the year in question, while the report ends with a financial statement. Among diseases we note 308 of simple xerosis with keratomalacia of which 4 were admitted to hospital for in-patient treatment. The superintendent is to be congratulated on the way in which, in spite of the difficulties of the times, the hospital has been running in full work.

CORRESPONDENCE

UNUSUAL CASES OF CONJUNCTIVITIS

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—By the quotation of certain phrases from the account of four unusual cases of conjunctivitis which I submitted in the October number of the British Journal of Ophthalmology, Major R. E. Wright suggests an attempt on my part to describe a new conjunctival disease. Such was not my intention, which would have been presumptuous indeed on so small a series.

I was merely concerned to report these cases, which I had found of interest, and to point out that though they differed considerably in clinical appearance each was monocular, chronic in course, and revealed similar non-specific changes on biopsy.

The list of conditions which Major Wright indicates may be included in Parinaud's oculo-glandular syndrome lends support to his view that this syndrome is liberal in its clinical confines; it would seem that such a label can add little information to a particular case.

I took as my guide to Verhoeff's leptothicosis conjunctivae his original paper published in 1918. As I pointed out, the special staining for the organism was not carried out but the clinical course and pathological findings for my cases were so dissimilar from the Author's description that I felt justified in excluding this diagnosis.

Major Wright mentioned that the first of my cases had been previously under his care and I would like to correct any implied discourtesy in not having acknowledged this fact. Any chronic recurrent disease in a service patient tends to gravitate to a number of Army Medical Centres. It happens that the four cases under discussion were seen by one or more colleagues, to have referred to each of whom would have burdened unnecessarily a paper of small scope.

Yours, etc.

E. F. KING.

Major, R.A.M.C.

January, 1943.

This correspondence is now closed—EDITORS.

THE VERTEBRATE EYE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—I have not seen the volume by Dr. Gordon Lynn Walls, "The Vertebrate Eye and its Adaptive Radiation," but I have read the review published in the December number of the British Journal of Ophthalmology.

In fairness to the author I hasten to challenge the following unwarranted comments made by the reviewer. The statement that "it is certainly the ciliary epithelium covering the ciliary folds and processes" which is the immediate source of the intra-ocular fluid (p. 371) is misleading in the light of recent research, and is indeed rather surprising as emanating from a comparative physiologist.

My personal acquaintance with the Vertebrate Eye is limited to mammalia. In this field and on this subject under discussion, the

most recent contribution, to my knowledge, is my paper, "The Mechanism of Aqueous Secretion in Mammalia," which appeared in the British Journal of Ophthalmology in January, 1941. Your reviewer has obviously not made himself acquainted with the demonstration presented there, for his criticisms and strictures are not only ill-judged but illuminated by prejudice and not by the light of reason.

I am, etc.,

THOMSON HENDERSON.

15, THE ROPEWALK,
NOTTINGHAM.
January 15, 1943.

MACULAR HOLE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—May I report another case similar to that described by Lieutenant Commander Pittar?

R. A., aged 13, attended a school clinic on Nov. 4, 1942, complaining of a black spot in his right vision. Vision was 6/12 6/5, the eyes emmetropic. There was oedema of the right macula. Thinking it might be an early central choroiditis I treated him with atropine and rest. The oedema gradually subsided, being replaced by an appearance of abnormal redness. About this time the boy's mother suggested that the trouble might be due to his having looked at the sun without protection.

On Dec. 30, there was a definite macular "hole" with clean cut edges, though vision was 6/18. This retention of fair vision is surprising, but one occasionally meets it in cases of old central choroiditis in which the ophthalmoscopic appearance suggests a total loss of central vision. No doubt it will deteriorate further.

Yours truly,

H. DOUGLAS SMART.

18, WIMPOLE STREET, W.1

SYMPATHETIC OPHTHALMITIS

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—On p. 572 of your Journal for December, 1942, a letter from Mr. T. Harrison Butler is published in which is described a case supposed to be one of sympathetic ophthalmitis. The interval of time between the date of excision of the exciting eye and that of the first signs of inflammation in the sympathising eye was

3½ months (April 10 to July 23). Improvement followed treatment by the end of August. In November recrudescence of inflammation occurred and glaucoma supervened. Excision of the eye was advised.

Surely this is a case above all others in which facts, if obtainable, are required. If enucleation of the eye has been carried out, I am sure that many of your readers would like to know the result of the histological examination. If this is unobtainable, I expect that many besides myself will prefer to regard "Mr. T. L., aged 69, a feeble, worn-looking man," whose doctor gave his opinion that he was "completely worn out and has been a heavy drinker" as the subject of irido-cyclitis with secondary glaucoma, due to some undefined cause other than sympathetic ophthalmitis. In support of this view, may I quote Mr. Harrison Butler's original article:—on p. 548 he says "before we can make a diagnosis (of sympathetic ophthalmitis) we must satisfy ourselves . . . . that there is no other extraneous cause for the inflammation in the sympathising eye."

I am, Sirs,

Yours faithfully,

HUMPHREY NEAME.

21, DEVONSHIRE PLACE, W.1
February 1, 1943.

OBITUARY

PERCIVAL JOHN HAY

OPHTHALMOLOGISTS throughout the world will read with regret of the death of Dr. P. J. Hay, of Sheffield.

Percival John Hay was born on June 28, 1875 at Basle, Switzerland. His father was John Hay, a Scotsman, who became Professor of English in Basle University, and his mother was from the Manchester district of Lancashire. Hay was educated at Basle and before commencing Medicine at Edinburgh he was for a year an undergraduate at Basle University. He graduated M.B., Ch.B. Edin. in 1901, and M.D. with high commendation three years later. He held Resident appointments at the Birmingham and Midland Ear and Throat Hospital and the Birmingham and Midland Eye Hospital, after which, in 1908 he started practice in Sheffield as an Ophthalmic Surgeon, and for a time acted as Clinical Assistant to the late Simeon Snell at the Sheffield Royal Infirmary. He was appointed Honorary Ophthalmic Surgeon to the Beckett's Hospital, Barnsley, in 1910, Honorary Ophthalmic Surgeon to the Sheffield Royal Hospital in 1916, and at about the same time, Ophthalmic Surgeon to the Sheffield Education Committee.

PERCIVAL J HAY

(1875—1943)

During the 1914-18 War he served as Captain in the R.A.M.C. and was for some time Ophthalmic Specialist to the Cambridge Hospital, Aldershot. At the commencement of the present hostilities he immediately went back into hard hospital practice in order to relieve a colleague who had been mobilized. There is no doubt that the extra work which he put in during the last three years contributed in no small measure to the illness which caused his death.

It was just before the outbreak of War in 1914 that Hay, in association with the late Gray Clegg, of Manchester, was instrumental in founding the North of England Ophthalmological Society; he became its Honorary Secretary and remained so until his death, combining the office with that of President in 1931-32. It was as Secretary of the North of England Society that Hay showed his genius for organisation. Regular Clinical Meetings were instituted at Manchester, Bradford, Leeds, Newcastle, Liverpool and Sheffield, and a first class Pathological service established. The outstanding success on such occasions as the joint meetings with the Midland Ophthalmological Society, the Scottish Ophthalmological Club, and the Royal Society of Medicine, was entirely due to his untiring efforts.

He was responsible for the annual lectures given to the North of England Society by men of the calibre of Van der Hoeve, Magitot, Gjessing, Von Rohr, Ronne, Goulden, Ballantyne, Duke-Elder, Traquair and Roaf. Perhaps his most outstanding success was the organisation of his famous tours—in 1935 it was Germany, Austria and Italy—in 1937 the Scandinavian countries, and in 1939 the U.S.A. and Canada, and had he lived he had other equally attractive plans maturing for future development. It was on these occasions that his ability as a linguist was so helpful and made all the difference to the smooth running of the programme; on one memorable occasion he was unexpectedly called upon to speak to a mixed audience in Sweden; he commenced in English, continued in French and German, and finished in Swedish. Also, one cannot forget the week-end which the Society spent in London as the guests of the Honorary Staff at Moorfields.

It can be truthfully said that Hay devoted his life to Ophthalmology. As Lecturer in Ophthalmology at the Sheffield University he was tireless in his efforts to improve the teaching facilities, both in the lecture room and at hospital, and he pressed hard for the founding of a Chair in Ophthalmology.

He was an indefatigable worker, hated slackness and had an uncanny instinct for picking out a genuine worker, to whom he would devote time and patience. He possessed a wide knowledge of ophthalmology and his medical and surgical colleagues rarely met him in consultation without picking up some new idea.

He contributed widely to ophthalmic literature and in 1929 he delivered the Richard Middlemore Lecture on "Astigmatism."

He was a Vice-President of the Ophthalmic Section of the Royal Society of Medicine; was a Past President of the local Division of the B.M.A., and a Past President of the Sheffield Medico-Chirurgical Society. In later years he achieved two of the highest honours which can come to a British Ophthalmologist—he became Master of the Oxford Ophthalmological Congress in 1939, and last year was elected President of the Ophthalmological Society of the United Kingdom. It will be a great disappointment to his ophthalmic colleagues that he was not spared to preside over the Society during the coming year.

Hay had many excellencies of character—industrious and careful in his work, with a vast store of scientific and clinical knowledge; wise in his counsels, and a perfect host with a sense of humour hidden behind a naturally shy disposition. He was a remarkably good photographer and very musical.

He leaves one daughter, Mrs. John Lane, his wife having predeceased him many years ago.

In spite of an outer reserve of character it was the privilege of many to know him well, and those who did, valued his friendship and cherish his memory.

Mr. W. H. Kiep writes:—

The North of England Ophthalmological Society has sustained an irreparable loss in the death of Percival J. Hay. He not only founded the Society but was very largely responsible for the smooth and successful running of it. I do not think any medical society has ever had a more zealous and kindly secretary. We all know that a secretary is largely responsible for the success or failure of a medical society. Percival Hay built up the Society stone by stone, and having first arranged clinical meetings at the various North of England cities, he later arranged for lecturers of distinction to come and deliver the "January Lectures." Later on still, he organised tours, the first being to Hungary and Italy, the second to the Northern capitals and the last to America. The success of these tours can be judged by the fact that members of almost all other ophthalmological societies availed themselves of the opportunity to participate in the tour.

The outstanding characteristic of Percival Hay was that he would spare himself no effort to make the meetings a success and he would spend endless time in drawing up agenda or even dinner plans in such a way as to secure the harmoniousness of the meeting. Anything that savoured of pungent criticism he sincerely deprecated, and was anxious that the members should all be on terms of friendship one with the other. Assuredly it may be said of him that he was a "gentle soul."

IVOR LLOYD TUCKETT

IVOR LLOYD TUCKETT died at his home in Cowes, I.O.W., on November 28, 1942. Born in 1873 he was educated at Marlborough and Trinity College, Cambridge, and took his medical course at University College Hospital where he was house physician. He took the "College" diplomas in 1898 and proceeded M.D.Cantab. in 1910. After leaving University College Hospital he was house surgeon at Moorfields; and next returned to Cambridge to act as senior demonstrator of Physiology and assistant to the Downing Professor of Medicine. After a few years he was elected Fellow of Trinity College and he was also a Fellow of University College, London. Tuckett joined the Ophthalmological Society in 1917. He contributed papers on intra-ocular foreign bodies to our earlier volumes. Much of his literary work was physiological, and he was the author of an important paper in the *Journal of Physiology* on the structure of non-medullated nerve fibres.

NOTES

Deaths

IT is with very great regret that we learn from Dr. Burton Chance, of Philadelphia, that the following outstanding American Ophthalmologists died towards the end of last year; Edward Jackson, Luther Peter and William Shoemaker. We hope to publish notices of each in a later number.

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Ophthalmological Society of the United Kingdom. Annual Congress, 1943

It is proposed to hold the Annual Congress on Friday, April 30, and Saturday, May 1, 1943, at the Royal Society of Medicine. The subjects chosen for discussion are:

1. Friday morning, "Thyrotoxicosis in its Relation to Ophthalmology," to be opened by Dr. Russell Brain and Mr. L. H. Savin.
2. Saturday morning, "The Scientific and Clinical Aspects of Night-Vision, opened by Professor W. J. B. Riddell and Air-Commodore P. C. Livingston, A.F.C.

There will be a joint clinical meeting with the Ophthalmological Section of the Royal Society of Medicine on Friday afternoon. Those wishing to show cases should notify Mr. A. J. B. Goldsmith, 15, Devonshire Place, W.1. Those who wish to read papers or to take part in the discussions should inform Mr. Frank Law, 36, Devonshire Place, W.1, as soon as possible. There will be no official dinner. Arrangements regarding hotel accommodation should be made by members in good time.

National Society for the Prevention of Blindness, Inc. Prize for Work on Glaucoma

THE National Society for the Prevention of Blindness announces that a prize of $250 will be awarded for the most valuable original paper during 1943 adding to the existing knowledge about the diagnosis of early glaucoma. The award will be made by the Society with the guidance of an ophthalmological committee composed of Dr. Arnold Knapp, Dr. Manuel Uribe Troncoso and Dr. Mark J. Schoenberg.

Papers may be presented by any ophthalmologist, student in ophthalmology or research worker of the Western Hemisphere and may be written in English, French, German, Italian, Spanish or Portuguese, but those written in the last four languages should be accompanied by a translation in English. Papers should be in the office of the National Society for the Prevention of Blindness, 1790, Broadway, New York City, by September 15, 1943.

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Oxford Ophthalmological Congress. Officers and Arrangements

THE date for the Oxford Ophthalmological Congress will be July 8 and 9 at Oxford. The Officers are: Master, P. G. Doyne. University Representative, Miss Ida Mann. Hon. Secretary and Treasurer, F. A. Anderson.

The Doyne Lecture will be delivered by Professor Adrian, of Cambridge, and the main discussion will be "Ophthalmic Education," to be opened by Sir John Parsons, Mr. R. H. Davenport and Dr. Derick Vail.

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Royal Society of Medicine

THE meetings of the Section of Ophthalmology during the session 1942-43 will be held as follows:—

Friday, March 12, at 2.30 p.m.
Friday, April 30, at 5.0 p.m.
Friday, June 11, at 5.0 p.m.

Cases and specimens will be shown half-an-hour before the advertised times of the meetings.

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Special Notice

WE are asked by the Ministry of Information to state that the fact that goods made of raw materials in short supply owing to war conditions are advertised in this journal should not be taken as an indication that they are necessarily available for export.